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The Role of an ABC Transporter as a Steroid Antagonist in Drosophila
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Introduction

Drosophila melanogaster are holometabolous insects that have several distinct life stages including larva and a winged adult. The larval stage is marked by a series of feeding and growth, while the adult stage is optimized for sexual reproduction and dispersal. The larval stage can be divided into three periods: larva, pupa, and imaginal discs (Figure 1). Larval growth – both internal and external – depends on specific signaling pathways controlled by a cholesterol-derived steroid, 20-hydroxyecdysone (20E). Although 20E is a systemic developmental signal, little is known about the molecular details of how different tissues respond to the hormone. We have been studying one gene induced by 20E in some target tissues (Figure 2). This gene, E23, encodes an ATP binding cassette (ABC) transporter protein that may function to limit hormone exposure in tissues where it is expressed.

Materials and Methods

To control the production of E23 within specific tissues, a powerful genetic system can be used: the UAS-Gal4 system (Figure 3). This system, which has been adopted from yeast, consists of two parts: the Expression Activating Sequence (UAS), and the GAL4 “driver” protein which binds to UAS. This allows for transcription and production of the UAS controlled E23 only in cells that express the Gal4-GAL4 construct. Figure 6 is a diagram of the procedure to assay for defects.

Results and Conclusion

Shown below is a graph comparing the control (UAS-E23/noGal4) and experimental (UAS-E23/Gal4-L14) larvae (Figure 7). The graph, from data following 150 larvae each, compares mortality by measuring living animals. On the right is a comparison of the third inactiv larvae (Figure 8). Ecdysone defects are indicated by a second set of mouth hooks, cuticular structures that are created during molting. If the larva cannot undergo ecdysis, the mouth hooks remain attached rather than falling off with the old cuticle.

Future Work

Along with observing the effects of UAS cells on the molting and ecdysis process, I will also be studying the effect of 20E suppression throughout the larval testis. The epidermal cells, which secrete the cuticular structures, can be controlled through another recently acquired another stock, AS-Cl.4. We hypothesize an overproduction of E23 in these cells would halt molting, preliminary data with this donor results in an extension of the larval L1 stage and lethality.

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References